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(FILE 'HOME' ENTERED AT 16:32:32 ON 11 JUL 2005)

FILE 'REGISTRY' ENTERED AT 16:32:49 ON 11 JUL 2005

L1 647 S (HEPATOCYTE GROWTH FACTOR)
L2 1 S 127651-39-2/RN

FILE 'CAPLUS' ENTERED AT 16:35:01 ON 11 JUL 2005
S L2/CN

FILE 'REGISTRY' ENTERED AT 16:35:07 ON 11 JUL 2005

FILE 'CAPLUS' ENTERED AT 16:35:07 ON 11 JUL 2005

L3 2 S L2
E (HEPATOCYTE GROWTH FACTOR)
L4 5161973 S E6
L5 5512 S (HEPATOCYTE GROWTH FACTOR)
L6 301 S L5 AND INTESTINE
L7 345 S L5 AND INTESTIN?
L8 129 S L7 NOT (EXPRESSION OR ANTIBODY)

=> d 18 110-129 bib,kwic

L8 ANSWER 110 OF 129 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1997:534189 CAPLUS
DN 127:200341
TI Induction of plasma **hepatocyte growth factor**
in acute colitis of mice
AU Matsuno, M.; Shiota, G.; Umeki, K.; Kawasaki, H.; Kojo, H.; Miura, K.
CS School Medicine, Tottori Univ., Yonago, 683, Japan
SO Inflammation Research (1997), 46(5), 166-167
CODEN: INREFB; ISSN: 1023-3830
PB Birkhaeuser
DT Journal
LA English
TI Induction of plasma **hepatocyte growth factor**
in acute colitis of mice
AB Acute colitis was induced in male FVB mice (about 25 g) by intrarectal
administration of 200 µl of 0.5% acetic acid. Blood plasma was taken
at 0, 12, 24, 36, 48, and 72 h after acetic acid treatment. Plasma
hepatocyte growth factor (HGF) was measured by
an enzyme immunoassay in duplicate. Plasma levels of HGF at 0, 12, 24,
36, 48, and 72 h after acetic acid administration were 0.31, 0.45, 0.46,
0.71, 0.82, and 0.44 ng/mL, resp. Increases in plasma HGF levels were
observed at 36 and 48 h after the treatment. Plasma HGF was induced by acute
inflammation of colonic tissues. Thus, HGF in blood may be 1 of the
non-specific markers of inflammation in mice.
ST **hepatocyte growth factor** colitis
IT **Intestine**, disease
(colitis; induction of plasma HGF in acute colitis of mice)
IT **Hepatocyte growth factor**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); BIOL (Biological study)
(induction of plasma HGF in acute colitis of mice)

L8 ANSWER 111 OF 129 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1997:499245 CAPLUS
DN 127:160579
TI Adoptive immunotherapy using clonal allogeneic cytotoxic T lymphocytes
(CTL)
IN Stauss, Hans Josef
PA RPMS Technology Ltd., UK; Stauss, Hans Josef
SO PCT Int. Appl., 85 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

272 INFLAMMATORIES
 231780 INFLAMMATORY
 (INFLAMMATORY OR INFLAMMATORIES)
 56279 BOWEL
 503 BOWELS
 56579 BOWEL
 (BOWEL OR BOWELS)
 1680345 DISEASE
 1603661 DISEASES
 2874229 DISEASE
 (DISEASE OR DISEASES)
 12665 INFLAMMATORY BOWEL DISEASE
 (INFLAMMATORY(W) BOWEL(W) DISEASE)
 91019 INTESTINE
 47408 INTESTINES
 126996 INTESTINE
 (INTESTINE OR INTESTINES)
 1106011 REVIEW/DT
 L4 308 (INFLAMMATORY BOWEL DISEASE) AND INTESTINE AND REVIEW/DT
 => s ((inflammatory bowel disease) and intestine)/ti and review/dt
 45859 INFLAMMATORY/TI
 71 INFLAMMATORIES/TI
 45902 INFLAMMATORY/TI
 ((INFLAMMATORY OR INFLAMMATORIES)/TI)
 21258 BOWEL/TI
 54 BOWELS/TI
 21311 BOWEL/TI
 ((BOWEL OR BOWELS)/TI)
 408123 DISEASE/TI
 134136 DISEASES/TI
 540255 DISEASE/TI
 ((DISEASE OR DISEASES)/TI)
 5923 INFLAMMATORY BOWEL DISEASE/TI
 ((INFLAMMATORY(W) BOWEL(W) DISEASE)/TI)
 26623 INTESTINE/TI
 1783 INTESTINES/TI
 28388 INTESTINE/TI
 ((INTESTINE OR INTESTINES)/TI)
 1106011 REVIEW/DT
 L5 6 ((INFLAMMATORY BOWEL DISEASE) AND INTESTINE)/TI AND REVIEW/DT

=> d bib,abs 1-6

L5 ANSWER 1 OF 6 MEDLINE on STN

Full Text
 AN 2004524981 MEDLINE
 DN PubMed ID: 15475760
 TI Mechanisms of natural tolerance in the **intestine**: implications for **inflammatory bowel disease**.
 AU Jump Robin L; Levine Alan D
 CS Department of Pathology, Case Western Reserve University School of Medicine, Cleveland, Ohio 44106-4952, USA.
 NC P01 DK-57756 (NIDDK)
 SO Inflammatory bowel diseases, (2004 Jul) 10 (4) 462-78. Ref: 202
 Journal code: 9508162. ISSN: 1078-0998.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 LA English
 FS Priority Journals
 EM 200411
 ED Entered STN: 20041022
 Last Updated on STN: 20041106
 Entered Medline: 20041106
 AB Tolerance, the regulated inability to respond to a specific immunologic stimulant, is a physiological event important to normal immune function. Just as loss of tolerance to self-proteins results in autoimmune diseases, we assert that loss of tolerance to commensal flora in the intestinal lumen leads to inflammatory bowel disease (IBD). Mechanisms through which the mucosal immune system establishes and remains hyporesponsive toward the presence of food proteins and commensal flora, which we define as natural tolerance, are discussed. In addition to the contributions by commensal flora, the innate host defense and the adaptive immune systems promote natural tolerance to sustain normal mucosal homeostasis. Understanding the molecular and cellular events that mediate natural tolerance will lead to more advanced insights into IBD pathogenesis and improved therapeutic options.

L5 ANSWER 2 OF 6 MEDLINE on STN

Full Text
 AN 2003043211 MEDLINE
 DN PubMed ID: 12552370
 TI [Barium examinations of the small **intestine** and the colon in **inflammatory bowel disease**].
 Konventionelle Dunn- und Dickdarmdiagnostik bei entzündlichen Darmerkrankungen.
 AU Antes G
 CS Abteilung für Radiologie, Klinikum Kempten-Oberallgäu gGmbH, Kempten..
 Gantes@gmx.de
 SO Der Radiologe, (2003 Jan) 43 (1) 9-16. Ref: 14
 Journal code: 0401257. ISSN: 0033-832X.
 CY Germany; Germany, Federal Republic of
 DT Journal; Article; (JOURNAL ARTICLE)